sent in to the department from a town where the infestation of cone-noses in a certain house was said to be particularly severe. The inhabitants had been bitten on several occasions, and reported itchings, swellings, and general nervous disturbances as results of the bites. The writer in this instance also felt no ill effects.

The validity of some of the reports concerning the serious effects of cone-nose bites can hardly be questioned, but they are probably frequently exaggerated. The finding of a big black bug over half an inch long in the house or in one's bed is likely to cause an "itchy" feeling, or to have a "paralyzing" effect on the finder, even without his having been bitten. It is also a well-known fact that some individuals are more sensitive than others to insect bites, poison oak and other irritants. A sensitive individual bitten by a cone-nose will probably show many, or perhaps all, of the symptoms reported, while a less sensitive person would not react at all.

The danger of cone-noses, however, should not be underestimated, as trypanosomiasis, various other diseases and secondary infections may be caused either by contamination or bites of these insects.

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THE RÔLE OF GLYCIN IN MUSCLE **METABOLISM**

The large group of myopathies, of which progressive muscular dystrophy is an outstanding example, has been looked upon for some little time as essentially a disturbance in muscle metabolism.

As early as 1870 it was known that certain myopathies were associated with a severe disturbance in creatinin excretion, as indicated by a diminished daily output.

In 1909, Levene and Kristeller 2 found in cases of progressive muscular dystrophy that there was not only a low creatinin, but a high creatin output, which was increased by a larger intake of protein in the diet. In other words, these patients had a lowered or diminished tolerance to creatin.

Recently a number of investigators have reported metabolic studies in this field with special reference to glycin administration. Kostakow and Slauck 8 have reached the conclusion that glycin, the simplest alpha amino-acid, plays a very important rôle in muscle physiology. Their studies have convinced these authors that in progressive muscular dystrophy the body has lost the ability to utilize creatin, regaining this capacity under the influence of glycin. Therefore, glycin is of benefit in muscular dystrophy and the authors think that the glycin treatment has far-reaching possibilities.

This work had been greatly stimulated by the efforts of Brand, Harris, Sandberg, and Ringer,4 who reported in 1929 that when glycin is fed to patients with progressive muscular dystrophy, an appreciable increase in creatin excretion takes place; indicating, in all their experiments, that there is a special and significant relationship of glycin to creatin metabolism.

Thomas, Milhorat, and Techner⁵ repeated these experiments, and fed glycin over prolonged periods of time to various patients with involvement of the muscular system; and they reported that the administration of this amino-acid had a marked therapeutic effect in some cases of progressive muscular dystrophy. In cases showing clinical improvement with treatment, there was an associated drop in creatin excretion with a rise in creatinin.

Remen,⁶ and Boothby,⁷ working with cases of myasthenia gravis, have shown encouraging results with the use of glycin, and glycin together with ephedrin.

Harris and Brand,8 in discussing their cases of myopathy after prolonged glycin administration, are fairly conservative in the estimation of their clinical results, but agree that this type of investigation has yielded information both interesting and extremely worth while.

The Lancet,9 in an editorial discussion on the muscular dystrophies, does not believe that this particular field of medicine is likely to be enlightened in the way of therapy from clinical studies alone, and that chemical investigation in these conditions is an uncultivated field which will repay any labor spent upon it. In agreement with Harris and Brand, "this statement is not quoted to minimize the important clinical contributions of the earlier investigators, but rather to indicate along what lines further progress probably lies."

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Local Anesthetic for Nose and Throat.-From a study of the use of the hydrochlorid of piperidinopropanedial diphenylurethane (diothane) as a local anesthetic for the nose and throat, Stitt concludes that the drug, if properly used, is a satisfactory local anesthetic to replace cocain for all routine uses in the nose and throat. It may be used in considerably lower concentrations than cocain, although the onset of anesthesia is somewhat slower. The resultant anesthesia is more lasting than that produced by cocain and frequently much more striking. Its action on the ear drum allows an almost painless paracentesis. In a series of several hundred cases in which hydrochlorid of piperidinopropanedial diphenylurethane has been used, there have been no signs of toxicity and the drug has not produced the undesirable reactions sometimes found with cocain. It is safe for routine use as a spray to relieve pain after tonsillectomy and is preferred for this purpose to acetylsalicylic acid.—Annals of Otolaryngology, Rhinology and Laryngology.

¹ Rosenthal, M.: Handbuch der Diagnostik und Therapie der Nerven-Krankheiten, Erlangen, 1870.
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3 Kostakow, S., and Slauck, A.: Glycine Treatment of Progressive Muscular Dystrophy. Deutsche Med. Wchnschr., 59:169 (Feb. 3), 1933.

⁴ Brand, E., Harris, M. M., Sandberg, M., and Ringer,
A. I.: Am. J. Physiol., 90:296, 1929.
5 Thomas, K., Milhorat, A. T., and Techner, F.: Ztschr.
f. Physiol. Chem., 205:93, 1932.
6 Remen, L.: Deutsche Ztschr. f. Nervenh., 128:66, 1932.
7 Boothby, W. M., and others: Proc. Staff Meet., Mayo Clinic, 7:557 and 737, 1932.
8 Harris, M. H., and Brand, E.: Metabolic and Therapeutic Studies in the Myopathies, J. A. M. A., 101:1047 (Sept. 30), 1933.
9 The Muscular Dystrophies: Editorial, Lancet, 2:1179, 1925.